FILE 'HOME' ENTERED AT 13:47:02 ON 16 OCT 2006

=> file reg `
COST IN U.S. DOLLARS SINCE FILE TOTAL . ENTRY SESSION FULL ESTIMATED COST 0.21 0.21

FILE 'REGISTRY' ENTERED AT 13:47:31 ON 16 OCT 2006

-> 0	amyloid/cn	
=> C E1	1	AMYLOHEPTAOSE/CN
E2	1	AMYLOHEXAOSE/CN
E3		AMYLOID/CN
E4	1	AMYLOID (CANDIDA ALBICANS)/CN
E5		AMYLOID (HUMAN CLONE 72HO4 FRAGMENT)/CN
E6	1	AMYLOID (HUMAN HEREDITARY RENAL AMYLOIDOSIS-ASSOCIATED)/CN
E7	1	AMYLOID (HUMAN P-COMPONENT)/CN
E8	1	AMYLOID (MESOCRICETUS AURATUS)/CN
E9	1	AMYLOID (MONKEY) PROTEIN A/CN
E10	1	AMYLOID (MOUSE CLONE ΛΜΙΑΡ-G2)/CN
E11	1	AMYLOID (MOUSE)/CN
E12	1	AMYLOID (PICHIA PINUS)/CN
=> e		
E13	17	AMYLOID (SACCHAROMYCES CEREVISIAE)/CN
E14	1	AMYLOID (SYNTHETIC CLONE 10)/CN
E15	1	AMYLOID (SYNTHETIC CLONE 12)/CN
E16	1	AMYLOID (SYNTHETIC CLONE 16)/CN
E17	1	AMYLOID (SYNTHETIC CLONE 17)/CN
E18	1	AMYLOID (SYNTHETIC CLONE 19)/CN
E19	1	AMYLOID (SYNTHETIC CLONE 23)/CN
E20	1	AMYLOID (SYNTHETIC CLONE 24)/CN
E21	1	AMYLOID (SYNTHETIC CLONE 4)/CN
E22	1	AMYLOID (SYNTHETIC CLONE 66)/CN
E23	1 .	AMYLOID (SYNTHETIC CLONE 68)/CN
E24	.1	AMYLOID (SYNTHETIC CLONE 69)/CN
_		
=> e	-	TAMES OF THE ASSESSMENT OF THE STATE OF THE
E25	1	AMYLOID (SYNTHETIC CLONE 7)/CN
E26	1	AMYLOID (SYNTHETIC CLONE 71)/CN
E27	1	AMYLOID (SYNTHETIC CLONE 75)/CN
E28	1.	AMYLOID (SYNTHETIC CLONE 78)/CN
E29	1	AMYLOID (SYNTHETIC CLONE 8)/CN
E30	1	AMYLOID (SYNTHETIC)/CN
E31	1	AMYLOID B (HUMAN 7-AMINO ACID N-TERMINAL FRAGMENT TRIPL
		E REPEAT) FUSION PROTEIN WITH TETANUS TOXOID (CLOSTRIDIUM TÉ
		TANI 820-844/947-967 FRAGMENT)/CN
E32	1	AMYLOID B (HUMAN 7-AMINO ACID N-TERMINAL FRAGMENT) FUSI
		ON PROTEIN WITH 307-319-HEMAGGLUTININ (INFLUENZA VIRUS) FUSI
		ON PROTEIN WITH ANTIGEN CS (CIRCUMSPOROZOITE) (MALARIA CIRCU
		MSPOROZOITE) FUSION/CN
E33	1	AMYLOID B 1-42/CN
E34	1	AMYLOID B PEPTIDE(1-40) (SYNTHETIC)/CN
E35	1	AMYLOID B-PEPTIDE-BINDING ALCOHOL DEHYDROGENASE/CN
E36	1	AMYLOID B-PROTEIN PRECURSOR (HUMAN C-TERMINAL FRAGMENT)
		/CN

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=> s e33-34 or e36
             1 "AMYLOID B 1-42"/CN
             1 "AMYLOID B PEPTIDE(1-40) (SYNTHETIC)"/CN
             1 "AMYLOID B-PROTEIN PRECURSOR (HUMAN C-TERMINAL FRAGMENT)"/C
L9
             3 ("AMYLOID B 1-42"/CN OR "AMYLOID B PEPTIDE(1-40) (SYNT
               HETIC) "/CN) OR "AMYLOID B-PROTEIN PRECURSOR (HUMAN C-TERMIN
              AL FRAGMENT) "/CN
=> file caplus
COST IN U.S. DOLLARS
                                                  SINCE FILE
                                                                   TOTAL
                                                       ENTRY
                                                                 SESSION
FULL ESTIMATED COST
                                                       15.16
                                                                   50.25
FILE 'CAPLUS' ENTERED AT 13:53:12 ON 16 OCT 2006
=> s 19/anst
          2189 L9
       1105363 ANST/RL
L10
            61 L9/ANST
                 (L9 (L) ANST/RL)
=> s 110 and pd<20021106
      22742485 PD<20021106
                 (PD<20021106)
L11
            25 L10 AND PD<20021106
=> s l11 and (mass spec?)
        937694 MASS
         81912 MASSES
        979393 MASS
                 (MASS OR MASSES)
       5351198 SPEC?
        290434 SP
          7668 SPS
        297726 SP
                 (SP OR SPS)
       5564761 SPEC?
                 (SPEC? OR SP)
        307183 MASS SPEC?
                 (MASS(W)SPEC?)
L12
             3 L11 AND (MASS SPEC?)
=> d bib abs 1-3
L12 ANSWER 1 OF 3 CAPLUS COPYRIGHT 2006 ACS on STN
AN
     1999:777514 CAPLUS Full-text
DN
     132:148708
     On-line high-performance liquid chromatography/mass
TI
     spectrometric investigation of amyloid-\beta peptide variants
     found in Alzheimer's disease
     Thompson, Andrew J.; Lim, Teck K.; Barrow, Colin J.
AU
     School of Chemistry, University of Melbourne, Parkville, 3052, Australia
CS
SO
     Rapid Communications in Mass Spectrometry (1999), 13(23),
     2348-2351
     CODEN: RCMSEF; ISSN: 0951-4198
PB
     John Wiley & Sons Ltd.
     Journal
DT
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- LA English
- AB A β peptides are the major components of amyloid deposits in Alzheimer's disease. The presence of N-terminally truncated A β variants in amyloid may be a critical factor in Alzheimer's disease pathogenesis. These A β variants are less soluble and more amyloidogenic than full-length A β , making their separation, purification and identification difficult. High-performance liquid chromatog. (HPLC) at elevated temps., coupled to electrospray ionization (ES) mass spectrometry (MS), enables rapid separation and identification of N-terminally truncated A β variants. This methodol. provides a potential tool for exploring the importance of these A β variants in both the pathogenesis and diagnosis of Alzheimer's disease.
- RE.CNT 22 THERE ARE 22 CITED REFERENCES AVAILABLE FOR THIS RECORD ALL CITATIONS AVAILABLE IN THE RE FORMAT
- L12 ANSWER 2 OF 3 CAPLUS COPYRIGHT 2006 ACS on STN
- AN 1997:21587 CAPLUS Full-text
- DN 126:130138
- TI The profile of soluble amyloid β protein in cultured cell media. Detection and quantification of amyloid β protein and variants by immunoprecipitation-mass spectrometry
- AU Wang, Rong; Sweeney, David; Gandy, Samuel E.; Sisodia, Sangram S.
- CS Lab. Mass Spectrometry, Rockefeller Univ., New York, NY, 10021, USA
- SO Journal of Biological Chemistry (1996), 271(50), 31894-31902 CODEN: JBCHA3; ISSN: 0021-9258
- PB American Society for Biochemistry and Molecular Biology
- DT Journal
- LA English
- To study the metabolism of amyloid β protein (A β) in Alzheimer's disease, the AB authors have developed a new approach for analyzing the profile of soluble Aß and its variants. In the present method, $A\beta$ and its variants are immunoisolated with $A\beta$ -specific monoclonal antibodies. The identities of the Aß variants are determined by measuring their mol. masses using matrixassisted laser desorption ionization time-of-flight mass spectrometry. The levels of $A\beta$ variants are determined by their relative peak intensities in mass spectrometric measurements by comparison with internal stds. of known identities and concns. The authors used this method to examine the Aß species in conditioned media of mouse neuroblastoma cells transfected with cDNAs encoding wild type or mutant human amyloid precursor protein. In addition to human $A\beta$ -(1-40) and $A\beta$ -(1-42), more than 40 different human $A\beta$ variants were identified. Endogenous murine Aß and its variants were also identified by this approach. The present approach is a new and sensitive method to characterize the profile of soluble $A\beta$ in conditioned media and biol. fluids. Furthermore, it allows direct measurement of each individual peptide in a peptide mixture and provides comprehensive information on the identity and concentration of $A\beta$ and $A\beta$ variants.
- RE.CNT 44 THERE ARE 44 CITED REFERENCES AVAILABLE FOR THIS RECORD ALL CITATIONS AVAILABLE IN THE RE FORMAT
- L12 ANSWER 3 OF 3 CAPLUS COPYRIGHT 2006 ACS on STN
- AN 1994:698351 CAPLUS Full-text
- DN 121:298351
- TI APP717 missense mutation affects the ratio of amyloid β protein species (Aβ1-42/43 and Aβ1-40) in familial Alzheimer's disease brain
- AU Tamaoka, Akira; Odaka, Asano; Ishibashi, Yoshihiro; Usami, Mihoko; Sahara, Naruhiko; Suzuki, Nobuhiro; Nukina, Nobuyuki; Mizusawa, Hidehiro; Shoji, Shin'ichiro; et al.

- CS Inst. Clinical Med., Univ. Tsukuba, Ibaraki, 305, Japan SO Journal of Biological Chemistry (1994), 269(52), 32721-4
 - CODEN: JBCHA3; ISSN: 0021-9258
 American Society for Biochemistry and Molecular Biology
- DT Journal

PB

- LA English
- We have biochem. purified $A\beta$ from brains of two unrelated familial Alzheimer's AB disease (FAD) pedigrees with the APP717 mutation (Val \rightarrow Ile) and from two sporadic Alzheimer's disease (AD) brains and characterized them by means of mass spectrometry and ELISA. We observed two types of amyloid β protein $(A\beta)$, the short-tail form $(A\beta1-40)$ and the longtail form $(A\beta1-42/43)$, in sporadic AD and FAD brains, and found that the ratio of the long-tail form of A β (A β 1-42/43) to total A β was increased in FAD brains. These in vivo results were confirmed in vitro using cultured cells transfected with three kinds of APP cDNAs bearing the APP717 mutations (Val \rightarrow Ile, Gly, or Phe). Taken together with the hypothesis that $A\beta 1-42/43$ functions as "seed" that increases the kinetics of amyloid fibril formation, we conclude that the APP717 missense mutation does not create new $A\beta$ species but promotes the increased accumulation of $A\beta 1-42/43$ in the brain, which results in the enhancement of amyloid fibril formation from soluble AB. These findings provide a causal relationship between this FAD genotype and the pathol. phenotype of $A\beta$ deposition and senile plaque formation.

=> 10g Y		
COST IN U.S. DOLLARS	SINCE FILE	TOTAL
	ENTRY	SESSION
FULL ESTIMATED COST	18.78	69.03
DISCOUNT AMOUNTS (FOR QUALIFYING ACCOUNTS)	SINCE FILE	TOTAL
	ENTRY '	SESSION
CA SUBSCRIBER PRICE .	-2.25	-2.25

STN INTERNATIONAL LOGOFF AT 13:56:49 ON 16 OCT 2006